A CASE OF ACUTE ASCENDING PARALYSIS IN A CHIMPANZEE

BY

S. ADLER

AND

E. J. CLARK

(*Sir Alfred Lewis Jones Research Laboratory, Freetown*)

(*Received for publication* 15 *November*, 1922)

A male chimpanzee (*Anthropopithecus troglodytes*), judged to be about four years of age, was captured by natives on the 3rd of September, 1922, near Blama, Sierra Leone.

The animal appeared healthy and showed no evidence of injury to the spine. On the evening of the 5th of September the animal was chloroformed in order to be caged, and was then sent down to Freetown. Very little chloroform was used and the animal quickly recovered from the anaesthetic.

On arrival in Freetown, on the 7th of September, two abrasions in the loins, due to the chafing of a rope, were observed; these quickly improved on the application of iodine.

On the 15th of September it was noticed that the animal refused to leave its cage and did not take food; on removal from the cage it was found that the lower limbs were completely paralysed and were colder than the rest of the body. The animal eagerly drank large quantities of milk and water, but refused solid food.

On the evening of the 17th of September the trunk and the upper limbs were completely paralysed; the face was cyanosed and the animal suffered from dyspnoea. Milk feeds were vomited. The muscles of the neck were not affected and the animal could move its head freely from side to side.

On the 18th of September 0·09 gms. of Novarsenobillon were administered intramuscularly into the thigh; there was no loss of

sensation and the animal moved its head vigorously from side to side and attempted to seize the hand of an assistant with its teeth. The animal's condition appeared to improve rapidly after the administration of Novarsenobillon; the cyanosis disappeared and the respiration improved, but it was still unable to swallow solid food and lived entirely on milk.

On the 19th of September the animal passed a solid motion, the first since the 14th of September. As the condition appeared improved after the injection of Novarsenobillon, a second dose of 0·09 gms. was administered on the 20th of September.

No change occurred until the 23rd of September, when general fibrillary twitchings affecting all the muscles of the body were noticed; these twitchings were controlled by an injection of one-eighth grain of morphia. They recurred on the 24th of September, on which day the animal died.

A post-mortem was performed almost immediately after death.

The liver was pale yellowish in colour, and on section showed marked fatty degeneration.

Central nervous system. The cerebro-spinal fluid was slightly turbid and contained a few polynuclear leucocytes. The surface of the brain and cord were congested. Pieces of the cord from the mid-dorsal and upper cervical regions, and pieces of the medulla and cerebral cortex from the motor region (upper and lower limb centres), were fixed in alcohol, embedded in paraffin and sectioned; others were sectioned without embedding. Sections were stained in toluidine and thionin blue and differentiated with alcohol. Eosin and methylene blue, Giemsa, Leishman and Ehrlich's haematoxylin were also used.

Microscopically, the following changes were noticed in the central nervous system:—

Many of the cells in the cord, medulla and cortex were normal and showed Nissl's granules. In the anterior and posterior horns, and in Clarke's column, a number of cells showed faintly staining protoplasm and absence of Nissl's granules, and the nucleus tended to be eccentric in some cells. Vacuolisation of the cell protoplasm was observed in a number of cells; the vacuoles varied in size from 2μ to 6μ, and from one to six were found in each cell. Single vacuoles were found in cells which did not show marked

degeneration, but were noted in large numbers only in cells where degeneration was advanced. The vessels were congested and small haemorrhages were found. Similar changes were noted in the medulla, where vacuolisation of degenerated nerve cells was more marked than in the spinal cord.

Sections of the motor cortex from the upper and lower limb centres showed engorgement of the capillaries. No haemorrhages were found and the gross cellular changes found in the medulla and cord were not seen.

Sections of peripheral nerves showed no pathological changes.

Cultures of the heart's blood were negative.

On the 18th of September 0·2 c.c. of the animal's blood were injected intraperitoneally into a *Cercopithecus campbelli* with negative result.

On the 24th of September, during the post-mortem, 3·5 c.c. of cerebro-spinal fluid were injected intraperitoneally into a *Cercopithecus campbelli*. No paralysis followed. The animal died on the 27th of October, 1922. Post-mortem examination revealed an abscess involving the whole of the upper lobe of the right lung. Smears showed the presence of a Gram-negative capsulated pneumobacillus and a Gram-negative coccus, which was isolated in pure culture.

Before the chimpanzee's illness it had shared a cage with three younger chimpanzees which remained healthy. This, in conjunction with the fact that injection of blood and cerebro-spinal fluid into *Cercopithecus campbelli* produced no paralysis, indicates that the condition was not one of acute anterior polyomyelitis.

The lapse of time between the administration of chloroform and the appearance of symptoms also indicates that delayed chloroform poisoning was not responsible for the condition. Professor Blacklock suggests that the arsenic administered may have contributed to the condition of the liver.

The case presents interest in its close resemblance to the course of acute ascending paralysis as described in human beings.